



ARDEX CE760

ARDEX (Ardex Australia)

Chemwatch: 9013-80

Version No: 2.1

Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Chemwatch Hazard Alert Code: 3

Initial Date: 22/04/2026

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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	ARDEX CE760
Chemical Name	Not Applicable
Synonyms	Not Available
Chemical formula	Not Applicable
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Use according to manufacturer's directions.
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Details of the manufacturer or importer of the safety data sheet

Registered company name	ARDEX (Ardex Australia)
Address	2 Buda Way Kempers Creek NSW 2147 Australia
Telephone	1300 788 780
Fax	1300 780 102
Website	www.ardexaustralia.com
Email	technical.services@ardexaustralia.com

Emergency telephone number

Association / Organisation	ARDEX (ARDEX Australia)	CHEMWATCH EMERGENCY RESPONSE (24/7)
Emergency telephone number(s)	1800 224 070 (Mon-Fri, 9am-5pm)	+61 1800 951 288 (ID#: 9013-80)
Other emergency telephone number(s)	Not Available	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

HAZARDOUS CHEMICAL. NON-DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Germ Cell Mutagenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H335	May cause respiratory irritation.
H341	Suspected of causing genetic defects.
H373	May cause damage to organs through prolonged or repeated exposure.

Precautionary statement(s) Prevention

P260	Do not breathe dust/fume.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P202	Do not handle until all safety precautions have been read and understood.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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No further product hazard information.

SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
14808-60-7.	30-60	<u>graded sand</u>
65997-15-1	10-30	<u>portland cement</u>
12004-14-7	10-30	<u>calcium aluminate sulfate</u>
7778-18-9	10-30	<u>calcium sulfate</u>
1327-36-2	1-5	<u>aluminosilicate</u>
14808-60-7	<0.1	<u>silica crystalline - quartz</u>
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

SECTION 4 First aid measures**Description of first aid measures**

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary.

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	<ul style="list-style-type: none"> ▶ Transport to hospital, or doctor, without delay.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

For acute or short-term repeated exposures to highly alkaline materials:

- ▶ Respiratory stress is uncommon but present occasionally because of soft tissue edema.
- ▶ Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
- ▶ Oxygen is given as indicated.
- ▶ The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
- ▶ Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

- ▶ Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

- ▶ Neutralising agents should never be given since exothermic heat reaction may compound injury.

* Catharsis and emesis are absolutely contra-indicated.

* Activated charcoal does not absorb alkali.

* Gastric lavage should not be used.

Supportive care involves the following:

- ▶ Withhold oral feedings initially.
- ▶ If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
- ▶ Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
- ▶ Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

- ▶ Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

- ▶ There is no restriction on the type of extinguisher which may be used.
- ▶ Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Non combustible. ▶ Not considered a significant fire risk, however containers may burn. <p>Decomposition may produce toxic fumes of:</p> <p>sulfur oxides (SO_x) silicon dioxide (SiO₂) metal oxides</p> <p>When aluminium oxide dust is dispersed in air, firefighters should wear protection against inhalation of dust particles, which can also contain hazardous substances from the fire absorbed on the alumina particles.</p> <p>May emit poisonous fumes. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up waste regularly and abnormal spills immediately. ▶ Avoid breathing dust and contact with skin and eyes. ▶ Wear protective clothing, gloves, safety glasses and dust respirator. ▶ Use dry clean up procedures and avoid generating dust. ▶ Vacuum up or sweep up. NOTE: Vacuum cleaner must be fitted with an exhaust micro filter (H-Class HEPA type) (consider explosion-proof machines designed to be grounded during storage and use). H-Class HEPA filtered industrial vacuum cleaners should NOT be used on wet materials or surfaces. ▶ Dampen with water to prevent dusting before sweeping. ▶ Place in suitable containers for disposal.
Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard.

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- ▶ Wear full body protective clothing with breathing apparatus.
- ▶ Prevent, by all means available, spillage from entering drains or water courses.
- ▶ Consider evacuation (or protect in place).
- ▶ No smoking, naked lights or ignition sources.
- ▶ Increase ventilation.
- ▶ Stop leak if safe to do so.
- ▶ Water spray or fog may be used to disperse / absorb vapour.
- ▶ Contain or absorb spill with sand, earth or vermiculite.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling

- ▶ Avoid skin contact, including inhalation.
- ▶ Wear protective clothing when risk of exposure occurs.
- ▶ Use in a well-ventilated area.
- ▶ Prevent concentration in hollows and sumps.
- ▶ **DO NOT enter confined spaces until atmosphere has been checked.**
- ▶ **DO NOT** allow material to come in direct contact with human skin or eyes.
- ▶ **DO NOT** allow material to come in contact with exposed food or food contact surfaces.
- ▶ Suitable PPE must be worn at all times.
- ▶ Avoid contact with incompatible materials.
- ▶ **When handling, DO NOT eat, drink or smoke.**
- ▶ Keep containers securely sealed when not in use.
- ▶ Avoid physical damage to containers.
- ▶ Always wash hands with soap and water after handling.
- ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.
- ▶ Use good occupational work practice.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

Other information

- ▶ Store in original containers.
 - ▶ Keep containers securely sealed.
 - ▶ Store in a cool, dry area protected from environmental extremes.
 - ▶ Store away from incompatible materials and foodstuff containers.
 - ▶ Protect containers against physical damage and check regularly for leaks.
 - ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- For major quantities:
- ▶ Consider storage in banded areas - ensure storage areas are isolated from sources of community water (including stormwater, ground water, lakes and streams).
 - ▶ Ensure that accidental discharge to air or water is the subject of a contingency disaster management plan; this may require consultation with local authorities.

Conditions for safe storage, including any incompatibilities

Suitable container

Multi-ply paper bag with sealed plastic liner or heavy gauge plastic bag.

NOTE: Bags should be stacked, blocked, interlocked, and limited in height so that they are stable and secure against sliding or collapse. Check that all containers are clearly labelled and free from leaks. Packing as recommended by manufacturer.

Storage incompatibility

Inorganic alkaline earth metal derivative.
Derivative of very electropositive metal.
For aluminas (aluminium oxide):
Incompatible with hot chlorinated rubber.
In the presence of chlorine trifluoride may react violently and ignite.
-May initiate explosive polymerisation of olefin oxides including ethylene oxide.
-Produces exothermic reaction above 200°C with halocarbons and an exothermic reaction at ambient temperatures with halocarbons in the presence of other metals.
-Produces exothermic reaction with oxygen difluoride.
-May form explosive mixture with oxygen difluoride.
-Forms explosive mixtures with sodium nitrate.
-Reacts vigorously with vinyl acetate.
Aluminium oxide is an amphoteric substance, meaning it can react with both acids and bases, such as hydrofluoric acid and sodium hydroxide, acting as an acid with a base and a base with an acid, neutralising the other and producing a salt.
Calcium oxide:

- ▶ reacts violently with water, evolving high quantities of heat
- ▶ reacts violently, with possible ignition or explosion, with acids, anilinium perchlorate, bromine pentafluoride, chlorine trifluoride, fluorine, hydrogen fluoride, hydrazine, hydrogen sulfide, hydrogen trisulfide, isopropyl isocyanide dichloride, light metals, lithium, magnesium, powdered aluminium, phosphorus, potassium, sulfur trioxide
- ▶ increase the explosive sensitivity of azides, nitroalkanes (e.g. nitroethane, nitromethane, 1-nitropropane etc.)
- ▶ is incompatible with boric acid, boron trifluoride, carbon dioxide, ethanol, halogens (such as fluorine), metal halides, phosphorus pentoxide, selenium oxychloride, sulfur dioxide and many organic materials

Calcium sulfate:

- ▶ reacts violently with reducing agents, acrolein, alcohols, chlorine trifluoride, diazomethane, ethers, fluorine, hydrazine, hydrazinium perchlorate, hydrogen peroxide, finely divided aluminium or magnesium, peroxyfuroic acid, red phosphorus, sodium acetylide
- ▶ sensitises most organic azides which are unstable shock- and heat- sensitive explosives
- ▶ may form explosive materials with 1,3-di(5-tetrazoly)triazene
- ▶ is incompatible with glycidol, isopropyl chlorocarbonate, nitrosyl perchlorate, sodium borohydride
- ▶ is hygroscopic; reacts with water to form gypsum and Plaster of Paris

For iron oxide (ferric oxide):

- ▶ Avoid storage with aluminium, calcium hypochlorite and ethylene oxide.
- ▶ Risk of explosion occurs following reaction with powdered aluminium, calcium silicide, ethylene oxide (polymerises), carbon monoxide, magnesium and perchlorates.
- ▶ Risk of ignition or formation of flammable gases or vapours occurs following reaction with carbides, for example caesium carbide, (produces heat), hydrogen sulfide, hydrogen peroxide (decomposes).

- ▶ An intimately powdered mixture with aluminium, usually ignited by magnesium ribbon, reacts with an intense exotherm to produce molten iron in the commercial "thermit" welding process
- ▶ **WARNING:** Avoid or control reaction with peroxides. All *transition metal* peroxides should be considered as potentially explosive. For example transition metal complexes of alkyl hydroperoxides may decompose explosively.
- ▶ The pi-complexes formed between chromium(0), vanadium(0) and other transition metals (haloarene-metal complexes) and mono- or poly-fluorobenzene show extreme sensitivity to heat and are explosive.
- ▶ Avoid reaction with borohydrides or cyanoborohydrides
- ▶ Metals and their oxides or salts may react violently with chlorine trifluoride and bromine trifluoride.
- ▶ These trifluorides are hypergolic oxidisers. They ignite on contact (without external source of heat or ignition) with recognised fuels - contact with these materials, following an ambient or slightly elevated temperature, is often violent and may produce ignition.
- ▶ The state of subdivision may affect the results.
- ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.
- ▶ Avoid contact with copper, aluminium and their alloys.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	graded sand	Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	graded sand	Silica - Crystalline: Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	graded sand	Silica - crystalline: Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Workers exposed to this chemical may require specific health monitoring (see regulations 368-378, Schedule 14 to the model WHS Regulations). The use, handling and storage of this chemical is subject to restriction or prohibition (see regulations 340, 380 - 384 and Schedule 10 to the model WHS Regulations).
Australia Exposure Standards	portland cement	Portland cement	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	portland cement	Portland cement (respirable dust)	1 mg/m3	Not Available	Not Available	Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	calcium sulfate	Calcium sulphate	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	calcium sulfate	Calcium sulfate (respirable)	1.5 mg/m3	Not Available	Not Available	Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	silica crystalline - quartz	Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	silica crystalline - quartz	Silica - Crystalline: Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) (Effective from 1 December 2026) - Appendix A - Workplace Exposure Limits	silica crystalline - quartz	Silica - crystalline: Quartz (respirable dust)	0.05 mg/m3	Not Available	Not Available	Workers exposed to this chemical may require specific health monitoring (see regulations 368-378, Schedule 14 to the model WHS Regulations). The use, handling and storage of this chemical is subject to restriction or prohibition (see regulations 340, 380 - 384 and Schedule 10 to the model WHS Regulations).

MATERIAL DATA

Exposure controls

Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

Employers may need to use multiple types of controls to prevent employee overexposure.

- ▶ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area.
- ▶ Work should be undertaken in an isolated system such as a "glove-box". Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system.

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	<ul style="list-style-type: none"> ▶ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▶ Open-vessel systems are prohibited. ▶ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▶ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▶ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▶ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▶ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▶ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.
<p>Individual protection measures, such as personal protective equipment</p>	
<p>Eye and face protection</p>	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].
<p>Skin protection</p>	<p>See Hand protection below</p>
<p>Hands/feet protection</p>	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> - frequency and duration of contact, - chemical resistance of glove material, - glove thickness and - dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> - When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. - Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> - Excellent when breakthrough time > 480 min - Good when breakthrough time > 20 min - Fair when breakthrough time < 20 min - Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> - Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. - Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Experience indicates that the following polymers are suitable as glove materials for protection against undissolved, dry solids, where abrasive particles are not present.</p> <ul style="list-style-type: none"> ▶ polychloroprene. ▶ nitrile rubber. ▶ butyl rubber. ▶ fluorocautchouc. ▶ polyvinyl chloride. <p>Gloves should be examined for wear and/ or degradation constantly.</p>
<p>Body protection</p>	<p>See Other protection below</p>
<p>Other protection</p>	<ul style="list-style-type: none"> ▶ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▶ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▶ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely.

- ▶ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood.
- ▶ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood.
- ▶ Overalls.
- ▶ P.V.C apron.
- ▶ Barrier cream.
- ▶ Skin cleansing cream.
- ▶ Eye wash unit.

Respiratory protection

Type -P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	P1 Air-line*	- -	PAPR-P1 -
up to 50 x ES	Air-line**	P2	PAPR-P2
up to 100 x ES	-	P3	-
		Air-line*	-
100+ x ES	-	Air-line**	PAPR-P3

* - Negative pressure demand ** - Continuous flow

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO₂), G = Agricultural chemicals, K = Ammonia(NH₃), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- Respirators may be necessary when engineering and administrative controls do not adequately prevent exposures.
- The decision to use respiratory protection should be based on professional judgment that takes into account toxicity information, exposure measurement data, and frequency and likelihood of the worker's exposure - ensure users are not subject to high thermal loads which may result in heat stress or distress due to personal protective equipment (powered, positive flow, full face apparatus may be an option).
- Published occupational exposure limits, where they exist, will assist in determining the adequacy of the selected respiratory protection. These may be government mandated or vendor recommended.
- Certified respirators will be useful for protecting workers from inhalation of particulates when properly selected and fit tested as part of a complete respiratory protection program.
- Where protection from nuisance levels of dusts are desired, use type N95 (US) or type P1 (EN143) dust masks. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU)
- Use approved positive flow mask if significant quantities of dust becomes airborne.
- Try to avoid creating dust conditions.

Where significant concentrations of the material are likely to enter the breathing zone, a Class P3 respirator may be required.

Class P3 particulate filters are used for protection against highly toxic or highly irritant particulates.

Filtration rate: Filters at least 99.95% of airborne particles

Suitable for:

- Relatively small particles generated by mechanical processes eg. grinding, cutting, sanding, drilling, sawing.
- Sub-micron thermally generated particles e.g. welding fumes, fertilizer and bushfire smoke.
- Biologically active airborne particles under specified infection control applications e.g. viruses, bacteria, COVID-19, SARS
- Highly toxic particles e.g. Organophosphate Insecticides, Radionuclides, Asbestos

Note: P3 Rating can only be achieved when used with a Full Face Respirator or Powered Air-Purifying Respirator (PAPR). If used with any other respirator, it will only provide filtration protection up to a P2 rating.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Powder; partly soluble in water.		
Physical state	Divided Solid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Available	Decomposition temperature (°C)	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Applicable
Initial boiling point and boiling range (°C)	Not Applicable	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Applicable
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Applicable	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Heat of Combustion (kJ/g)	Not Available	Ignition Distance (cm)	Not Available
Flame Height (cm)	Not Available	Flame Duration (s)	Not Available

Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	Enclosed Space Ignition Deflagration Density (g/m3)	Not Available
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SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	There is sufficient evidence to classify this material as mutagenic
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	There is sufficient evidence to classify this material as toxic to specific organs through single exposure
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	<p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of dusts, generated by the material during the course of normal handling, may be damaging to the health of the individual. Levels above 10 ug/m³ of suspended inorganic sulfates in the air may cause an excess risk of asthmatic attacks in susceptible persons. Inhalation may result in chrome ulcers or sores of nasal mucosa and lung damage.</p> <p>Persons with impaired respiratory function, airway diseases and conditions such as emphysema or chronic bronchitis, may incur further disability if excessive concentrations of particulate are inhaled.</p> <p>If prior damage to the circulatory or nervous systems has occurred or if kidney damage has been sustained, proper screenings should be conducted on individuals who may be exposed to further risk if handling and use of the material result in excessive exposures.</p> <p>Effects on lungs are significantly enhanced in the presence of respirable particles. Overexposure to respirable dust may produce wheezing, coughing and breathing difficulties leading to or symptomatic of impaired respiratory function.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Compared with other metals, the calcium ion and most calcium compounds have low toxicity. Acute calcium poisoning is rare, and difficult to achieve unless calcium compounds are administered intravenously or taken in high doses over a prolonged period..</p> <p>Excessive consumption of calcium carbonate antacids/dietary supplements over a period of weeks or months can cause milk-alkali syndrome, with symptoms ranging from hypercalcaemia to potentially fatal renal failure.</p> <p>A study investigating the effects of personal calcium supplement use on cardiovascular risk found a modestly increased risk of cardiovascular events, particularly myocardial infarction in postmenopausal women. In contrast, the authors of a 2013 literature review concluded that the benefits of calcium supplementation, such as on bone health, appear to outweigh any risk calcium supplementation may theoretically pose to the cardiovascular health.</p> <p>Excessive calcium supplementation can be detrimental to cardiovascular health, especially in men.</p> <p>Oral calcium supplements diminish the absorption of thyroxine when taken within four to six hours of each other. Thus, people taking both calcium and thyroxine run the risk of inadequate thyroid hormone replacement and thence hypothyroidism if they take them simultaneously or near-simultaneously.</p> <p>Chromate salts are corrosive because of their oxidising potency and produce tissue injury similar to acid burns. Ingestion may produce violent gastroenteritis, severe circulatory collapse and toxic nephritis. Peripheral vascular shock may also ensue.</p> <p>Acute toxic responses to aluminium are confined to the more soluble forms.</p> <p>Sulfate salts are poorly absorbed from the gastro-intestinal tract but because of osmotic activity are able to draw water from the lumen to produce diarrhoea (purging). Sulfate ion usually has little toxicological potential.</p> <p>Not normally a hazard due to the physical form of product. The material is a physical irritant to the gastro-intestinal tract</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Contact with aluminas (aluminium oxides) may produce a form of irritant dermatitis accompanied by pruritus.</p> <p>Though considered non-harmful, slight irritation may result from contact because of the abrasive nature of the aluminium oxide particles.</p>

	<p>Four students received severe hand burns whilst making moulds of their hands with dental plaster substituted for Plaster of Paris. The dental plaster known as "Stone" was a special form of calcium sulfate hemihydrate containing alpha-hemihydrate crystals that provide high compression strength to the moulds. Beta-hemihydrate (normal Plaster of Paris) does not cause skin burns in similar circumstances. Skin contact may result in severe irritation particularly to broken skin. Ulceration known as "chrome ulcers" may develop. Chrome ulcers and skin cancer are significantly related.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	<p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p>
Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Strong evidence exists that the substance may cause irreversible but non-lethal mutagenic effects following a single exposure. Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers. Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Chronic exposure to aluminas (aluminium oxides) of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. Epidemiologic surveys have indicated an excess of nonmalignant respiratory disease in workers exposed to aluminum oxide during abrasives production.</p> <p>Very fine Al₂O₃ powder was not fibrogenic in rats, guinea pigs, or hamsters when inhaled for 6 to 12 months and sacrificed at periods up to 12 months following the last exposure.</p> <p>When hydrated aluminas were injected intratracheally, they produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C.</p> <p>The weight of evidence suggests that catalytically active alumina and the large surface area aluminas can induce lung fibrosis (aluminosis) in experimental animals, but only when given by the intra-tracheal route. The pertinence of such experiments in relation to workplace exposure is doubtful especially since it has been demonstrated that the most reactive of the aluminas (i.e. the chi and gamma forms), when given by inhalation, are non-fibrogenic in experimental animals. However rats exposed by inhalation to refractory aluminium fibre showed mild fibrosis and possibly carcinogenic effects indicating that fibrous aluminas might exhibit different toxicology to non-fibrous forms. Aluminium oxide fibres administered by the intrapleural route produce clear evidence of carcinogenicity.</p> <p>Saffil fibre an artificially produced form alumina fibre used as refractories, consists of over 95% alumina, 3-4 % silica. Animal tests for fibrogenic, carcinogenic potential and oral toxicity have included in-vitro, intraperitoneal injection, intrapleural injection, inhalation, and feeding. The fibre has generally been inactive in animal studies. Also studies of Saffil dust clouds show very low respirable fraction.</p> <p>There is general agreement that particle size determines that the degree of pathogenicity (the ability of a micro-organism to produce infectious disease) of elementary aluminium, or its oxides or hydroxides when they occur as dusts, fumes or vapours. Only those particles small enough to enter the alveoli (sub 5 um) are able to produce pathogenic effects in the lungs.</p> <p>Red blood cells and rabbit alveolar macrophages exposed to calcium silicate insulation materials in vitro showed haemolysis in one study but not in another. Both studies showed the substance to be more cytotoxic than titanium dioxide but less toxic than asbestos.</p> <p>In a small cohort mortality study of workers in a wollastonite quarry, the observed number of deaths from all cancers combined and lung cancer were lower than expected. Wollastonite is a calcium inosilicate mineral (CaSiO₃). In some cases, small amounts of iron (Fe), and manganese (Mn), and lesser amounts of magnesium (Mg) substitute for calcium (Ca) in the mineral formulae (e.g., rhodonite)</p> <p>In an inhalation study in rats no increase in tumour incidence was observed but the number of fibres with lengths exceeding 5 um and a diameter of less than 3 um was relatively low. Four grades of wollastonite of different fibre size were tested for carcinogenicity in one experiment in rats by intrapleural implantation. There was no information on the purity of the four samples used. A slight increase in the incidence of pleural sarcomas was observed with three grades, all of which contained fibres greater than 4 um in length and less than 0.5 um in diameter.</p> <p>In two studies by intraperitoneal injection in rats using wollastonite with median fibre lengths of 8.1 um and 5.6 um respectively, no intra-abdominal tumours were found.</p> <p>Evidence from wollastonite miners suggests that occupational exposure can cause impaired respiratory function and pneumoconiosis. However animal studies have demonstrated that wollastonite fibres have low biopersistence and induce a transient inflammatory response compared to various forms of asbestos. A two-year inhalation study in rats at one dose showed no significant inflammation or fibrosis</p> <p>Occupational exposure to aluminium compounds may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnoea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibrosis with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic.</p> <p>Because aluminium competes with calcium for absorption, increased amounts of dietary aluminium may contribute to the reduced skeletal mineralisation (osteopenia) observed in preterm infants and infants with growth retardation. In very high doses, aluminium can cause neurotoxicity, and is associated with altered function of the blood-brain barrier. A small percentage of people are allergic to aluminium and experience contact dermatitis, digestive disorders, vomiting or other symptoms upon contact or ingestion of products containing aluminium, such as deodorants or antacids. In those without allergies, aluminium is not as toxic as heavy metals, but there is evidence of some toxicity if it is consumed in excessive amounts. Although the use of aluminium cookware has not been shown to lead to aluminium toxicity in general, excessive consumption of antacids containing aluminium compounds and excessive use of aluminium-containing antiperspirants provide more significant exposure levels. Studies have shown that consumption of acidic foods or liquids with aluminium significantly increases aluminium absorption, and maltol has been shown to increase the accumulation of aluminium in nervous and osseous tissue. Furthermore, aluminium increases oestrogen-related gene expression in human breast cancer cells cultured in the laboratory. These salts' estrogen-like effects have led to their classification as a metalloestrogen. Some researchers have expressed concerns that the aluminium in antiperspirants may increase the risk of breast cancer.</p> <p>After absorption, aluminium distributes to all tissues in animals and humans and accumulates in some, in particular bone. The main carrier of the aluminium ion in plasma is the iron binding protein, transferrin. Aluminium can enter the brain and reach the placenta and foetus. Aluminium may persist for a very long time in various organs and tissues before it is excreted in the urine. Although retention times for aluminium appear to be longer in humans than in rodents, there is little information allowing extrapolation from rodents to the humans. At high levels of exposure, some aluminium compounds may produce DNA damage in vitro and in vivo via indirect mechanisms. The database on carcinogenicity of aluminium compounds is limited. No indication of any carcinogenic potential was obtained in mice given aluminium potassium sulphate at high levels in the diet.</p>

Aluminium has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high concentrations of aluminium. It has been suggested that aluminium is implicated in the aetiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these hypotheses remain controversial. Several compounds containing aluminium have the potential to produce neurotoxicity (mice, rats) and to affect the male reproductive system (dogs). In addition, after maternal exposure they have shown embryotoxicity (mice) and have affected the developing nervous system in the offspring (mice, rats). The available studies have a number of limitations and do not allow any dose-response relationships to be established. The combined evidence from several studies in mice, rats and dogs that used dietary administration of aluminium compounds produce lowest-observed-adverse-effect levels (LOAELs) for effects on neurotoxicity, testes, embryotoxicity, and the developing nervous system of 52, 75, 100, and 50 mg aluminium/kg bw/day, respectively. Similarly, the lowest no-observed-adverse-effect levels (NOAELs) for effects on these endpoints were reported at 30, 27, 100, and for effects on the developing nervous system, between 10 and 42 mg aluminium/kg bw per day, respectively. Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations. Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro-transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA).

Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - *Chemistry in Australia, August 1995*]

the main target organs of aluminum are the central nervous system and bone. Aluminum binds with dietary phosphorus and impairs gastrointestinal absorption of phosphorus. The decreased phosphate body burden results in osteomalacia (softening of the bones due to defective bone mineralization) and rickets. Aluminum's neurotoxicity is believed to involve several mechanisms. Changes in cytoskeletal protein functions as a results of altered phosphorylation, proteolysis, transport, and synthesis are believed to be one cause. Aluminum may induce neurobehavioral effects by affecting permeability of the blood-brain barrier, cholinergic activity, signal transduction pathways, lipid peroxidation, and impair neuronal glutamate nitric oxide-cyclic GMP pathway, as well as interfere with metabolism of essential trace elements because of similar coordination chemistries and consequent competitive interactions. It has been suggested that aluminum's interaction with estrogen receptors, but studies have not been able to establish a clear link between aluminum and increased risk of breast cancer). Certain aluminum salts induce immune responses by activating inflammasomes.

Cement contact dermatitis (CCD) may occur when contact shows an allergic response, which may progress to sensitisation. Sensitisation is due to soluble chromates (chromate compounds) present in trace amounts in some cements and cement products. Soluble chromates readily penetrate intact skin. Cement dermatitis can be characterised by fissures, eczematous rash, dystrophic nails, and dry skin; acute contact with highly alkaline mixtures may cause localised necrosis.

Cement eczema may be due to chromium in feed stocks or contamination from materials of construction used in processing the cement. Sensitisation to chromium may be the leading cause of nickel and cobalt sensitivity and the high alkalinity of cement is an important factor in cement dermatoses [ILO].

Repeated, prolonged severe inhalation exposure may cause pulmonary oedema and rarely, pulmonary fibrosis. Workers may also suffer from dust-induced bronchitis with chronic bronchitis reported in 17% of a group occupationally exposed to high dust levels.

Respiratory symptoms and ventilatory function were studied in a group of 591 male Portland cement workers employed in four Taiwanese cement plants, with at least 5 years of exposure (1). This group had a significantly lowered mean forced vital capacity (FCV), forced expiratory volume at 1 s (FEV1) and forced expiratory flows after exhalation of 50% and 75% of the vital capacity (FEF50, FEF75). The data suggests that occupational exposure to Portland cement dust may lead to a higher incidence of chronic respiratory symptoms and a reduction of ventilatory capacity.

Chun-Yuh et al; *Journal of Toxicology and Environmental Health* 49: 581-588, 1996

High blood concentrations of calcium ion may give rise to vasodilation and depress cardiac function leading to hypotension and syncope. Calcium ions enhance the effects of digitalis on the heart and may precipitate digitalis intoxication. Calcium salts also reduce the absorption of tetracyclines

In neonates calcification of soft-tissue has been observed following therapeutic administration.

Some studies show that large quantities of calcium intake can cause hypercalcaemia, which can in turn lead to renal failure. Renal failure can occur within hours or days or, alternatively, settles gradually, evolving over several years until it reaches terminal stages. Similarly, acute renal failure can also develop into chronic forms of the disease.

Hypercalcaemia conditions can be associated with normal or reduced calcium serum levels, as the body tends to maintain a balanced metabolism of the mineral, known as the compensation phase. When there is a slight increase in the concentration of ions in the blood, calcium excretion markedly increases, while intestinal absorption decreases. After kidney damage has set in, a loss of calcium may occur, thereby decreasing the serum concentration.

Serum protein levels may decrease as a result of proteinuria in cases of renal complications. Proteinuria is an indicator of kidney disease and represents an independent risk factor for the progression of such a condition. Increased serum creatinine levels may represent an important parameter, given that kidney diseases are associated with increased serum creatinine levels. When renal pathology occurs, a progressive loss of glomerular filtration begins, resulting in increased plasma creatinine concentrations. During the course of kidney failure, discrete, but constant, increments in plasma creatinine levels occur.

Renal disease with albuminuria may also be the cause of hypoalbuminemia in patients with liver disease. In cases of established liver damage, increased calcium urinary excretion may occur. Therefore, a similar increase may cause the decline in serum calcium levels in the current study.

Overexposure to the breathable dust may cause coughing, wheezing, difficulty in breathing and impaired lung function. Chronic symptoms may include decreased vital lung capacity and chest infections. Repeated exposures in the workplace to high levels of fine-divided dusts may produce a condition known as pneumoconiosis, which is the lodgement of any inhaled dusts in the lung, irrespective of the effect. This is particularly true when a significant number of particles less than 0.5 microns (1/50000 inch) are present. Lung shadows are seen in the X-ray. Symptoms of pneumoconiosis may include a progressive dry cough, shortness of breath on exertion, increased chest expansion, weakness and weight loss. As the disease progresses, the cough produces stringy phlegm, vital capacity decreases further, and shortness of breath becomes more severe. Other signs or symptoms include changed breath sounds, reduced oxygen uptake during exercise, emphysema and rarely, pneumothorax (air in the lung cavity).

Removing workers from the possibility of further exposure to dust generally stops the progress of lung abnormalities. When there is high potential for worker exposure, examinations at regular period with emphasis on lung function should be performed.

Inhaling dust over an extended number of years may cause pneumoconiosis, which is the accumulation of dusts in the lungs and the subsequent tissue reaction. This may or may not be reversible.

Chronic excessive iron exposure has been associated with haemosiderosis and consequent possible damage to the liver and pancreas. Haemosiderin is a golden-brown insoluble protein produced by phagocytic digestion of haematin (an iron-based pigment). Haemosiderin is found in most tissues, especially in the liver, in the form of granules. Other sites of haemosiderin deposition include the pancreas and skin. A related condition, haemochromatosis, which involves a disorder of metabolism of these deposits, may produce cirrhosis of the liver, diabetes, and bronze pigmentation of the skin - heart failure may eventually occur.

Such exposure may also produce conjunctivitis, choroiditis, retinitis (both inflammatory conditions involving the eye) and siderosis of tissues if iron remains in these tissues. Siderosis is a form of pneumoconiosis produced by iron dusts. Siderosis also includes discoloration of organs, excess circulating iron and degeneration of the retina, lens and uvea as a result of the deposition of intraocular iron. Siderosis might also involve the lungs - involvement rarely develops before ten years of regular exposure. Often there is an accompanying inflammatory reaction of the bronchi. Permanent scarring of the lungs does not normally occur.

High levels of iron may raise the risk of cancer. This concern stems from the theory that iron causes oxidative damage to tissues and organs by generating highly reactive chemicals, called free radicals, which subsequently react with DNA. Cells may be disrupted and may become cancerous. People whose genetic disposition prevents them from keeping tight control over iron (e.g. those with the inherited disorder, haemochromatosis) may be at increased risk.

Iron overload in men may lead to diabetes, arthritis, liver cancer, heart irregularities and problems with other organs as iron builds up.

[K. Schmidt, New Scientist, No. 1919 pp.11-12, 2nd April, 1994]

Chromium(III) is considered an essential trace nutrient serving as a component of the "glucose tolerance factor" and a cofactor for insulin action. High concentrations of chromium are also found in RNA. Trivalent chromium is the most common form found in nature.

Chronic inhalation of trivalent chromium compounds produces irritation of the bronchus and lungs, dystrophic changes to the liver and kidney, pulmonary oedema, and adverse effects on macrophages. Intratracheal administration of chromium(III) oxide, in rats, increased the incidence of sarcomas, and tumors and reticulum cell sarcomas of the lung. There is inadequate evidence of carcinogenicity of chromium(III) compounds in experimental animals and humans (IARC).

Chronic exposure to hexavalent chromium compounds reportedly produces skin, eye and respiratory tract irritation, yellowing of the eyes and skin, allergic skin and respiratory reactions, diminished sense of smell and taste, blood disorders, liver and kidney damage, digestive disorders and lung damage. There is sufficient evidence of carcinogenicity of chromium(VI) compounds in experimental animals and humans to confirm these as Class 1 carcinogens (IARC).

Exposure to chromium during chrome production and in the chrome pigment industry is associated with cancer of the respiratory tract. A slight increase in gastrointestinal cancer following exposure to chromium compounds has also been reported. The greatest risk is attributed to exposure to acid-soluble, water-insoluble hexavalent chromium which occurs in roasting and refining processes. Animal studies support the idea that the most potent carcinogenic compounds are the slightly soluble hexavalent compounds. The cells are more active in the uptake of the hexavalent forms compared to trivalent forms and this may explain the difference in occupational effect. It is the trivalent form, however, which is metabolically active and binds with nucleic acid within the cell suggesting that chromium mutagenesis first requires biotransformation of the hexavalent form by reduction.

Hexavalent chromes produce chronic ulceration of skin surfaces (quite independent of other hypersensitivity reactions exhibited by the skin).

Water-soluble chromium(VI) compounds come close to the top of any published "hit list" of contact allergens (eczematogens) producing positive results in 4 to 10% of tested individuals. On the other hand only chromium(III) compounds can bind to high molecular weight carriers such as proteins to form a complete allergen (such as a hapten). Chromium(VI) compounds cannot. It is assumed that reduction must take place for such compounds to manifest any contact sensitivity. The apparent contradiction that chromium(VI) salts cause allergies to chromium(III) compounds but that allergy to chromium(III) compounds is difficult to demonstrate is accounted for by the different solubilities and skin penetration of these compounds. Water-soluble chromium(VI) salts penetrate the horny layer of the skin more readily than chromium(III) compounds which are bound by cross-linking in the horny layer ("tanning", as for leather) and therefore do not reach the cells involved in antigen processing.

Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.

Legend: 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

PORTLAND CEMENT

The following information refers to contact allergens as a group and may not be specific to this product.

Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

CALCIUM ALUMINATE SULFATE

for calcium:

Toxicity from calcium is not common because the gastrointestinal tract normally limits the amount of calcium absorbed. Therefore, short-term intake of large amounts of calcium does not generally produce any ill effects aside from **constipation** and an increased risk of kidney stones. However, more severe toxicity can occur when excess calcium is ingested over long periods, or when calcium is combined with increased amounts of vitamin D, which increases calcium absorption. Calcium toxicity is also sometimes found after excessive intravenous administration of calcium. Toxicity is manifested by abnormal deposition of calcium in tissues and by elevated blood calcium levels (hypercalcaemia). However, hypercalcaemia is often due to other causes, such as abnormally high amounts of parathyroid hormone (PTH). Usually, under these circumstances, bone density is lost and the resulting hypercalcaemia can cause kidney stones and abdominal pain.

Continued...

Some cancers can also cause hypercalcaemia, either by secreting abnormal proteins that act like PTH or by invading and killing bone cells causing them to release calcium. Very high levels of calcium can result in appetite loss, nausea, vomiting, abdominal pain, confusion, seizures, and even coma.

for calcium chloride:

Acute toxicity: The acute oral toxicity of calcium chloride is low: LD50 in mice is 1940-2045 mg/kg bw, 3798-4179 mg/kg bw in rats, and 500-1000 mg/kg bw in rabbits. The acute oral toxicity is attributed to the severe irritating property of the original substance or its high-concentration solutions to the gastrointestinal tract. In humans, however, acute oral toxicity is rare because large single doses induce nausea and vomiting. The dermal acute toxicity is negligible: LD50 in rabbits >5000 mg/kg bw. No significant change was found by gross necropsy examination except skin lesions at or near the site of administration. Hypercalcaemia may occur only when there exists other factors that alter calcium homeostasis, such as renal inefficiency and primary hyperthyroidism.

Irritation/corrosiveness studies conducted under OECD test guidelines indicate that calcium chloride is not/slightly irritating to skin but severely irritating to eyes of rabbits. Prolonged exposure and application of moistened material or concentrated solutions resulted in considerable skin irritation, however. Irritating effect of the substance was observed in human skin injuries caused by incidental contact with the substance or its high-concentration solutions.

Repeat dose toxicity: A limited oral repeated dose toxicity study shows no adverse effect of calcium chloride on rats fed on 1000-2000 mg/kg bw/day for 12 months. Calcium and chloride are both essential nutrients for humans and a daily intake of more than 1000 mg each of the ions is recommended. The establishment of the ADI for calcium chloride has not been deemed necessary by JECFA (Joint FAO/WHO Expert Committee on Food Additives)

Genotoxicity: Genetic toxicity of calcium chloride was negative in the bacterial mutation tests and the mammalian chromosome aberration test.

Reproductive and developmental toxicity: No reproductive toxicity study has been reported. A developmental toxicity study equivalent to an OECD Guideline study, on the other hand, reveals no toxic effects on dams or foetuses at doses up to 189 mg/kg bw/day (mice), 176 mg/kg bw/day (rats) and 169 mg/kg bw/day (rabbits).

CALCIUM SULFATE

Gypsum (calcium sulfate dihydrate) is a skin, eye, mucous membrane, and respiratory system irritant. Early studies of gypsum miners did not relate pneumoconiosis with chronic exposure to gypsum. Other studies in humans (as well as animals) showed no lung fibrosis produced by natural dusts of calcium sulfate except in the presence of silica. However, a series of studies reported chronic nonspecific respiratory diseases in gypsum industry workers in Gacki, Poland.

Unlike other fibers, gypsum is very soluble in the body; its half-life in the lungs has been estimated as minutes. In four healthy men receiving calcium supplementation with calcium sulfate (CaSO₄·1/2H₂O) (200 or 220 mg) for 22 days, an average absorption of 28.3% was reported. Several feeding studies in pigs on the bioavailability of calcium in calcium supplements, including gypsum, have been conducted. The bioavailability of calcium in gypsum was similar to that for calcitic limestone, oyster shell flour, marble dust, and aragonite, ranging from 85 to 102%. In mice, the i.p. and intragastric LD50 values were 6200 and 4704 mg/kg, respectively, for phosphogypsum (98% CaSO₄·H₂O). For Plaster of Paris, the values were 4415 and 5824, respectively. In rats, an intragastric LD50 of 9934 mg/kg was reported for phosphogypsum.

Repeat dose toxicity: In a study of 241 underground male workers employed in four gypsum mines in Nottinghamshire and Sussex for a year (November 1976-December 1977), results of chest X-rays, lung function tests, and respiratory systems suggested an association of the observed lung shadows with the higher quartz content in dust rather than to gypsum; the small round opacities in the lungs were characteristic of silica exposure.

Prophylactic examinations of workers in a gypsum extraction and production plant (dust concentration exceeded TLV 2.5- to 10-fold) reported no risk of pneumoconiosis due to gypsum exposure, while another study of gypsum manufacturing plant workers reported that chronic occupational exposure to gypsum dust had resulted in pulmonary ventilatory defect of the restrictive form.

Three cases of idiopathic interstitial pneumonia with multiple bullae throughout the lungs were seen in Japanese schoolteachers (lifetime occupation) exposed to chalk; 2/3 of the chalk was made from gypsum and small amounts of silica and other minerals.

In rats exposed to an aerosol of anhydrous calcium sulfate fibers (15 mg/m³) or a combination of milled and fibrous calcium sulfate (60 mg/m³) six hours per day, five days per week for three weeks, gypsum dust was quickly cleared from the lungs of via dissolution and mechanisms of particle clearance.

In guinea pigs given intraperitoneal (i.p.) injections of gypsum (doses not provided), gypsum was absorbed followed by the dissolution of gypsum in surrounding tissues. In another study, after i.p. injection of gypsum (2 cm³ of a 5 or 10% suspension in saline) into guinea pigs, which were sacrificed at intervals up to 180 days, most of the dust was found distributed in the peritoneum of the anterior abdominal wall. Gypsum dust produced irregular and clustered nodules, which decreased in size over time.

Direct administration of WTC PM2.5 [mostly composed of calcium-based compounds, including calcium sulfate (gypsum) and calcium carbonate (calcite)] (10, 32, or 100 µg) into the airways of mice produced mild to moderate lung inflammation and airway hyperresponsiveness at the high dose. [It was noted that WTC PM2.5 is composed of many chemical species and that their interactions may be related with development of airway hyperresponsiveness.] In female SPF Wistar rats intratracheally (i.t.) instilled with anhydrite dust (35 mg) and sacrificed three months later, an increase in total lipid or hydroxyproline content in the lungs was not observed compared to controls.

In inhalation (nose-only) experiments in which male F344 rats were exposed to calcium sulfate fiber aerosols (100 mg/m³) for six hours per day, five days per week for three weeks, there were no effects on the number of macrophages per alveolus, bronchoalveolar lavage fluid (BALF) protein concentration, or BALF g-glutamyl transpeptidase activity (g-GT). Following three weeks of recovery, nonprotein thiol levels (NPSH), mainly glutathione, were increased in animals. In follow-up experiments, rats were exposed to an aerosol of anhydrous calcium sulfate fibers (15 mg/m³) or a combination of milled and fibrous calcium sulfate (60 mg/m³) for the same duration. Calcium levels in the lungs were similar to those of controls; however, gypsum fibers were detected in the lungs of treated animals. Significant increases in NPSH levels in BALF were observed in rats killed immediately after exposure at both doses and in recovery group animals at the higher dose. At 15 mg/m³, almost all NPSH was lost in macrophages from all treated animals (including those in recovery), but a significant decrease in extracellular g-GT activity was seen only in recovery group animals. Overall, the findings were "considered to be non-pathological local effects due to physical factors related to the shape of the gypsum fibers and not to calcium sulphate per se."

Intratracheal administration of man-made calcium sulfate fiber (2.0 mg) once per week for five weeks resulted in no deaths or significant body weight changes in female Syrian hamsters compared to controls.

Inflammation (specifically, chronic alveolitis with macrophage and neutrophil aggregation) was observed in the lung.

In guinea pigs, inhalation of calcined gypsum dust (1.6 x 10⁴ particles/mL) for 44 hours per week in 5.5 days for two years, followed with or without a recovery period of up to 22 months, produced only minor effects in the lungs. There were 12 of 21 deaths over the entire experimental period. These were due to pneumonia or other pulmonary lesions; however, no significant gross signs of pulmonary disease or nodular or diffuse pneumoconiosis became significant. Beginning near 11 months, pigmentation and atelectasis were seen. During the recovery period, four of ten guinea pigs died; two died of pneumonia. Pigmentation continued in most animals but not atelectasis. Low-grade chronic inflammation, occurring in the first two months, also disappeared.

Mercury emissions controls on coal-fired power plants have increased the likelihood of the presence of mercury in synthetic gypsum formed in wet flue gas desulfurisation (FGD) systems and the finished wallboard produced from the FGD gypsum. In a study at a commercial wallboard plant, the raw FGD gypsum, the product stucco (beta form of CaSO₄·1/2H₂O), and the finished dry wallboard each contained about 1 µg Hg/g dry weight. Total mercury loss from the original FGD gypsum content was about 0.045 g Hg/ton dry gypsum processed

Synergistic/Antagonistic Effects: In rats, i.t. administration of anhydrite (5-35 mg) successively and simultaneously with quartz reduced the toxic effect of quartz in lung tissue. This protective effect on quartz toxicity was also seen in guinea pigs; calcined gypsum dust prevented or hindered the development of fibrosis. Natural anhydrite, however, increased the fibrogenic effect of cadmium sulfide in rats. Additionally, calcined gypsum dust had a stimulatory effect on experimental tuberculosis in guinea pigs.

Cytotoxicity: In Syrian hamster embryo cells, gypsum (up to 10 µg/cm²) did not induce apoptosis. Negative results were also found in mouse peritoneal macrophages (tested at 150 µg/mL gypsum dust) and in Chinese hamster lung V79-4 cells (tested up to 100 µg/mL).

Carcinogenicity: In female Sprague-Dawley rats, i.p. injection of natural anhydrite dusts from German coal mines (doses not provided) induced granulomas; whether gypsum was the causal factor was not established. In Wistar rats, four i.p. injections of gypsum (25 mg each) induced abdominal cavity tumours, mostly sarcomatous mesothelioma, in 5% of animals; first tumour was seen at 546 days. In a subsequent experiment using the same procedure, female Wistar rats exhibited the first tumour at 579 days after the last injection. Mean survival of the tumour-bearing rats (5.7% of test group) was 583 days, while mean survival of the test group was 587 days. Tumour types seen were a sarcoma having cellular polymorphism, a carcinoma, and a reticulosarcoma.

Intratracheal administration of man-made calcium sulfate fiber (2.0 mg) once per week for five weeks produced tumours in three of 20 female Syrian hamsters observed two years later. An anaplastic carcinoma was found in the heart, and one dark cell carcinoma was seen in

	<p>the kidney. Two tumours of unspecified types were observed in the rib.</p> <p>In guinea pigs, inhalation of gypsum (doses not provided) for 24 months produced no lung tumours.</p> <p>In rats, i.t. administration of gypsum (doses not provided in abstract) from FGD for up to 18 months produced no arterial blood gas changes or indications of secondary heart damage as compared to controls.</p> <p>In another study, a single i.t. dose (25 mg) of flue gas gypsum dust did not produce a pathological reaction when observed for up to 18 months. There were also no signs of developing granuloma of fibrosis of the lungs. Lead quickly accumulated in the femur after injection but was eliminated during the observation period. In the Ames test, the flue gas gypsum dust was negative.</p> <p>Genotoxicity: Calcium sulfate (up to 2.5%) was negative in Salmonella typhimurium strains TA1535, TA1537, and TA1538 and in Saccharomyces cerevisiae strain D4 with and without metabolic activation.</p> <p>Developmental toxicity: In pregnant mice, rats, and rabbits, daily oral administration of calcium sulfate (16-1600 mg/kg bw) beginning on gestation day 6 up to 18 produced no effects on maternal body weights, maternal or foetal survival, or nidation (embryo implantation); developmental effects were also not seen.</p>
ALUMINOSILICATE	Some aluminosilicates (eg. kyanite) have been named as equivocal tumorigens (RTECS criteria).
SILICA CRYSTALLINE - QUARTZ	<p>WARNING: For inhalation exposure <u>ONLY</u>: This substance has been classified by the IARC as Group 1: CARCINOGENIC TO HUMANS</p> <p>The International Agency for Research on Cancer (IARC) has classified occupational exposures to respirable (<5 µm) crystalline silica as being carcinogenic to humans. This classification is based on what IARC considered sufficient evidence from epidemiological studies of humans for the carcinogenicity of inhaled silica in the forms of quartz and cristobalite. Crystalline silica is also known to cause silicosis, a non-cancerous lung disease.</p> <p>Intermittent exposure produces; focal fibrosis, (pneumoconiosis), cough, dyspnoea, liver tumours.</p> <p>* Millions of particles per cubic foot (based on impinger samples counted by light field techniques).</p> <p>NOTE : the physical nature of quartz in the product determines whether it is likely to present a chronic health problem. To be a hazard the material must enter the breathing zone as respirable particles.</p>
GRADED SAND & PORTLAND CEMENT & CALCIUM ALUMINATE SULFATE & ALUMINOSILICATE	No significant acute toxicological data identified in literature search.
PORTLAND CEMENT & CALCIUM SULFATE	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

Acute Toxicity	✗	Carcinogenicity	✗
Skin Irritation/Corrosion	✓	Reproductivity	✗
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	✓
Mutagenicity	✓	Aspiration Hazard	✗

Legend: ✗ – Data either not available or does not fill the criteria for classification
 ✓ – Data available to make classification

SECTION 12 Ecological information

Toxicity					
	Endpoint	Test Duration (hr)	Species	Value	Source
ARDEX CE760	Not Available	Not Available	Not Available	Not Available	Not Available
graded sand	Not Available	Not Available	Not Available	Not Available	Not Available
portland cement	Not Available	Not Available	Not Available	Not Available	Not Available
calcium aluminate sulfate	EC50	72h	Algae or other aquatic plants	4.8mg/l	2
	EC50	48h	Crustacea	6.8mg/l	2
	EC10(ECx)	72h	Algae or other aquatic plants	2.3mg/l	2
	LC50	96h	Fish	>83mg/l	2
calcium sulfate	EC50	72h	Algae or other aquatic plants	>79mg/l	2
	EC50	96h	Algae or other aquatic plants	3200mg/L	4
	NOEC(ECx)	0.25h	Fish	75mg/l	4
	LC50	96h	Fish	>79mg/l	2

aluminosilicate	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	410mg/l	2
	EC50	48h	Crustacea	>10000mg/l	2
	EC10(ECx)	72h	Algae or other aquatic plants	33mg/l	2

silica crystalline - quartz	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
calcium sulfate	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
calcium sulfate	LOW (BCF = 3.162)

Mobility in soil

Ingredient	Mobility
calcium sulfate	LOW (Log KOC = 6.124)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible or consult manufacturer for recycling options. ▶ Consult State Land Waste Management Authority for disposal. ▶ Bury residue in an authorised landfill. ▶ Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.7. Maritime transport in bulk according to IMO instruments

14.7.1. Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

14.7.2. Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
graded sand	Not Applicable
portland cement	Not Applicable
calcium aluminate sulfate	Not Applicable
calcium sulfate	Not Applicable
aluminosilicate	Not Applicable
silica crystalline - quartz	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
graded sand	Not Applicable
portland cement	Not Applicable
calcium aluminate sulfate	Not Applicable
calcium sulfate	Not Applicable

Continued...

Product name	Ship Type
aluminosilicate	Not Applicable
silica crystalline - quartz	Not Applicable

SECTION 15 Regulatory information

Safety, health and environmental regulations / legislation specific for the substance or mixture

graded sand is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australian Inventory of Industrial Chemicals (AIIC)
 Chemical Footprint Project - Chemicals of High Concern List

portland cement is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

calcium aluminate sulfate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

calcium sulfate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

aluminosilicate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
 International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

silica crystalline - quartz is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
 Australia Model Work Health and Safety Regulations - Schedule 10 - Table 14.1 Hazardous chemicals (other than lead) requiring health monitoring
 Australian Inventory of Industrial Chemicals (AIIC)
 Chemical Footprint Project - Chemicals of High Concern List
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
 International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 1: Carcinogenic to humans

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (graded sand; portland cement; calcium aluminate sulfate; calcium sulfate; aluminosilicate; silica crystalline - quartz)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (portland cement; calcium aluminate sulfate; aluminosilicate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (portland cement; calcium aluminate sulfate)
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (calcium aluminate sulfate; aluminosilicate)
Vietnam - NCI	Yes
Russia - FBEPH	No (calcium aluminate sulfate)
UAE - Control List (Banned/Restricted Substances)	No (graded sand; portland cement; calcium aluminate sulfate; aluminosilicate; silica crystalline - quartz)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	22/04/2026
Initial Date	22/04/2026

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

- ▶ PC - TWA: Permissible Concentration-Time Weighted Average
- ▶ PC - STEL: Permissible Concentration-Short Term Exposure Limit
- ▶ IARC: International Agency for Research on Cancer
- ▶ ACGIH: American Conference of Governmental Industrial Hygienists
- ▶ STEL: Short Term Exposure Limit
- ▶ TEEL: Temporary Emergency Exposure Limit,
- ▶ IDLH: Immediately Dangerous to Life or Health Concentrations
- ▶ ES: Exposure Standard
- ▶ OSF: Odour Safety Factor
- ▶ NOAEL: No Observed Adverse Effect Level
- ▶ LOAEL: Lowest Observed Adverse Effect Level
- ▶ TLV: Threshold Limit Value
- ▶ LOD: Limit Of Detection
- ▶ OTV: Odour Threshold Value
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration
- ▶ MARPOL: International Convention for the Prevention of Pollution from Ships
- ▶ IMSBC: International Maritime Solid Bulk Cargoes Code
- ▶ IGC: International Gas Carrier Code
- ▶ IBC: International Bulk Chemical Code

- ▶ AIIC: Australian Inventory of Industrial Chemicals
- ▶ DSL: Domestic Substances List
- ▶ NDSL: Non-Domestic Substances List
- ▶ IECSC: Inventory of Existing Chemical Substance in China
- ▶ EINECS: European INventory of Existing Commercial chemical Substances
- ▶ ELINCS: European List of Notified Chemical Substances
- ▶ NLP: No-Longer Polymers
- ▶ ENCS: Existing and New Chemical Substances Inventory
- ▶ KECI: Korea Existing Chemicals Inventory
- ▶ NZIoC: New Zealand Inventory of Chemicals
- ▶ PICCS: Philippine Inventory of Chemicals and Chemical Substances
- ▶ TSCA: Toxic Substances Control Act
- ▶ TCSI: Taiwan Chemical Substance Inventory
- ▶ INSQ: Inventario Nacional de Sustancias Químicas
- ▶ NCI: National Chemical Inventory
- ▶ FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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